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Reflexión

Anosmia in Covid19: a bradykinin based etiopathological explaination

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Abstract

Anosmia is a common and unexplained symptom in Covid19 infection. According to some authors Covid19 infection could provoke an increase in bradykinin levels. As it has been described, olfactory bulb neuroblasts express bradykinin receptors that are the resposibles for their cell fate. This fact could explain Covid19 anosmia as a desestrcturation of the stablished conections in olfactory bulb due to an abnormal neuroblast differentiation.

Keywords: Anosmia, Covid19, SARS - CoV- 2, bradykinin, neuroblast.

Anosmia is described in patients with Covid19 in a percentage of 50%. However, its cause is currently unclear. The objective of this article is to establish a possible etiopathological cause for the anosmia described in Covid19.

According to an etiopathogenic hypothesis of Covid19 induced respiratory distress (1), this viral infection would cause an increase in bradykinin levels both at the pulmonary and blood levels. This bradykinin would be related to the respiratory distress caused by this pathogen (non-cardiogenic pulmonary edema due to vasodilation).

Our hypothesis is that bradykinin could be related to the anosmia produced by this pathogen considering that neural stem cells (neuroblasts), that are very abundant in the olfactory bulb, express receptors for bradykinin (2). Moreover, studies carried out with Icatibant (3), an inhibitor of bradykinin receptors, in tumor cells of various origins, reveals that inhibition of tumor growth is observed only in SK-N-SH cell line (neuroblastoma) in vitro, cells intimately related to the neuroblast of the olfactory bulb.

As it is well known, the olfactory bulb is one of the few places in the adult organism where neural progenitors (neuroblasts) are found (4). As it is described by various authors, bradykinin is capable of increasing neuroblast migration, dendrite formation, and thus neuronal maturation (5), (6).

In Covid19, elevated bradykinin levels could act at the level of the olfactory bulb neuroblasts, altering the interconnections already established between them and modifying olfactory bulb neuronal tissue architecture, causing the patient to experience cacosmia and anosmia until these new interconnections begin to stabilize

after viral infection ceases.

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